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Health and Environmental Effects Profile for Dinitrophenols

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Cincinnati, OH

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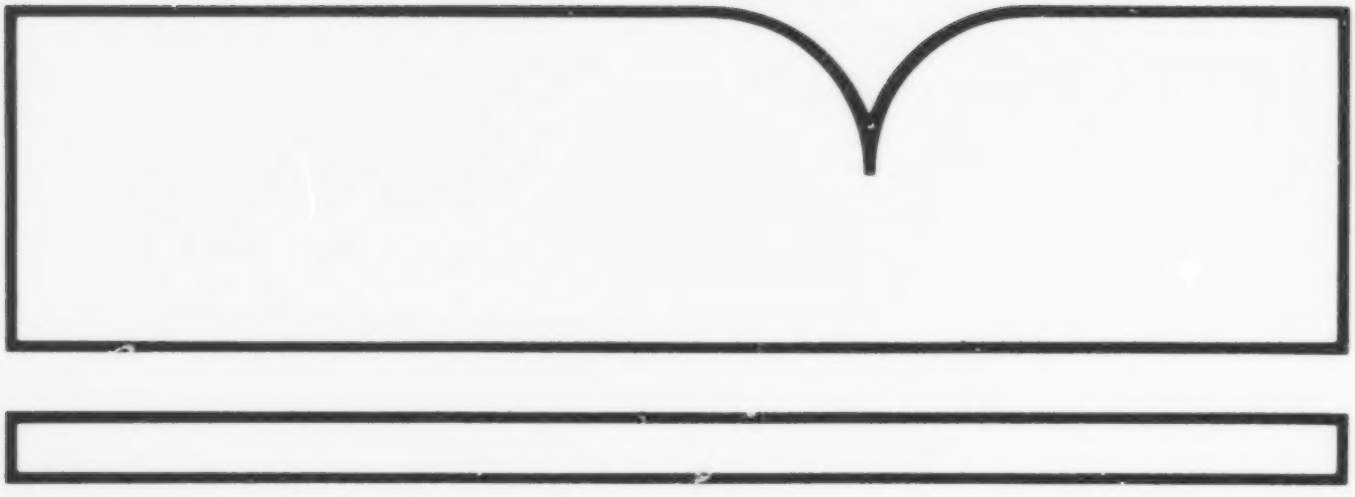

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR DINITROPHENOLS

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
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U.S. ENVIRONMENTAL PROTECTION AGENCY
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16. ABSTRACT

The Health and Environmental Effects Profile for 2,4-Dinitrophenol was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for other Dinitrophenol isomers.

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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LIST OF ABBREVIATIONS

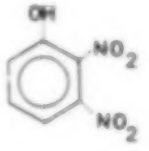
BCF	Bioconcentration factor
EC_{50}	Median effective concentration
i.p.	Intraperitoneal
K_a	Acid dissociation constant
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
MLD	Minimum lethal dose
NOEL	No-observed-effect level
NTP	National Toxicology Program
ppb	Parts per billion
TSCA	Toxic Substances Control Act

1. INTRODUCTION

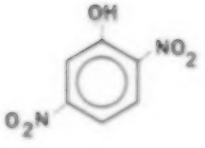
1.1. STRUCTURE AND CAS NUMBER

Dinitrophenol, a commercial product containing the 2,3-, 2,4- and 2,6-dinitrophenol isomers (Hawley, 1981; U.S. EPA/NIH, 1983a), is also known as dinitrophenol solution (NIOSH, 1983). 2,6-Dinitrophenol is also known as o-dinitrophenol, β-dinitrophenol, and DNP (trade name) (U.S. EPA/NIH, 1983a). 3,4-Dinitrophenol is also known as Δ-dinitrophenol and 4,5-dinitrophenol, and 3,5-dinitrophenol is known as e-dinitrophenol (U.S. EPA/NIH, 1983b). No synonyms were found for 2,3- or 2,5-dinitrophenol. The molecular formula of the dinitrophenol isomers is $C_6H_4N_2O_5$ and the molecular weight is 184.12 (NIOSH, 1983). This profile discusses the health and environmental effects of all isomers except the 2,4-dinitrophenol isomer. The structures of the selected dinitrophenols are given below:

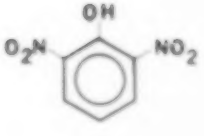
2,3-dinitrophenol:



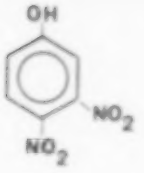
2,5-dinitrophenol:



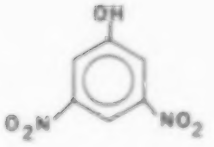
2,6-dinitrophenol:



3,4-dinitrophenol:



3,5-dinitrophenol:



The Chemical Abstracts Service (CAS) Registry Numbers are 25550-58-7 for dinitrophenol, 66-56-8 for 2,3-dinitrophenol, 321-71-5 for 2,5-dinitrophenol, 573-56-8 for 2,6-dinitrophenol, 577-71-9 for 3,4-dinitrophenol and 586-11-8 for 3,5-dinitrophenol.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Dinitrophenol is a commercial mixture composed of 2,3-, 2,4- and 2,6-dinitrophenol (Hawley, 1981; U.S. EPA/NIH, 1983a). Dinitrophenol exists as yellow crystals and is soluble in alcohol, ether, benzene and chloroform. Dinitrophenol is slightly soluble in water (quantitative data not specified) (Hawley, 1981).

2,3-Dinitrophenol exists as yellow monoclinic crystals (von Oettingen, 1941). Some of the physical properties of 2,3-dinitrophenol are given below:

Specific gravity:	1.681 (Hawley, 1981)
Melting point:	144°C (Hawley, 1981)
Solubility in water at 35-36°C:	2200 mg/l (Harvey, 1959)
K_a at 25°C:	1.3×10^{-5} (Harvey, 1959)

2,5-Dinitrophenol exists as yellow monoclinic crystals (von Oettingen, 1941). Some of the physical properties of 2,5-dinitrophenol are given below:

Melting point:	104°C (Harvey, 1959)
Solubility in water at 35-36°C:	680 mg/ℓ (Harvey, 1959)
K_a at 25°C:	7×10^{-4} (Harvey, 1959)

2,6-Dinitrophenol exists as yellow rhombic crystals (von Oettingen, 1941). Some of the physical properties of 2,6-dinitrophenol are given below:

Melting point:	63°C (Hawley, 1981)
Solubility in water at 35-36°C:	420 mg/ℓ (Harvey, 1959)
K_a at 25°C:	2.7×10^{-4} (Harvey, 1959)
Vapor density:	6.35 (U.S. EPA/NIH, 1983a)

3,4-Dinitrophenol exists as triclinic colorless or pale brown crystals (von Oettingen, 1941). Some of the physical properties of 3,4-dinitrophenol are given below:

Specific gravity (temperature not specified):	1.672 (Harvey, 1959)
Melting point:	134°C (Harvey, 1959)
Solubility in water at 35-36°C:	2300 mg/ℓ (Harvey, 1959)
K_a at 25°C:	4.3×10^{-5} (Harvey, 1959)

3,5-Dinitrophenol exists as colorless monoclinic crystals (von Oettingen, 1941). Some the physical properties of 3,5-dinitrophenol are given below:

Specific gravity (temperature not specified):	1.702 (Harvey, 1959)
Melting point:	122-123°C (Harvey, 1959)
Solubility in water at 35-36°C:	1600 mg/ℓ (Harvey, 1959)
K_a at 25°C:	2.1×10^{-4} (Harvey, 1959)

1.3. PRODUCTION DATA

Dinitrophenol is prepared by either heating phenol with dilute sulfuric acid, cooling the product, and then nitrating while keeping the temperature below 50°C, or by nitrating with a mixed acid under careful temperature control (Hawley, 1981). 2,3-, 2,5- and 3,4-Dinitrophenol are prepared by nitration of m-nitrophenol. 2,6-Dinitrophenol is prepared by sulfonation and nitration of o-nitrophenol. 3,5-Dinitrophenol is prepared by the replacement of one nitro group by methoxyl in sym-trinitrobenzene and demethylation of the dinitroanisole by anhydrous aluminum chloride (Harvey, 1959).

None of the selected dinitrophenol isomers are listed in the TSCA Inventory. 2,6-Dinitrophenol is produced by American Hoechst Corp., Industrial Chemicals Division, Organic Intermediates Dept., North Hollywood, CA (U.S. EPA/NIH, 1983a). Neither production sites nor production volumes for any of the other selected dinitrophenol isomers were located in the available literature.

The selected dinitrophenol isomers also may be produced in substantial quantity as by-products in the synthesis of 2,4-dinitrophenol by way of 1-chloro-2,4-dinitrobenzene. Production volumes for 1-chloro-2,4-dinitrobenzene were reported as 100,000-1,000,000 pounds by Pfister Chemical, Inc., Ridgefield, NJ, in 1977 (production volumes for six alternate production sites were not reported) (U.S. EPA/NIH, 1983c) and 5.3-8.5 million pounds from 1959-1968 (USITC, 1959-1968).

1.4. USE DATA

Dinitrophenols (unspecified) have a wide variety of biocidal action, and are used as insecticides, acaricides, herbicides and fungicides (Kirk-Othmer, 1971). Dinitrophenols (unspecified) are also used in the manufacture of styrene as inhibitors in the purification stills to reduce polymerization (Kirk-Othmer, 1971). Dinitrophenol (commercial mixture) and 2,6-dinitrophenol are used in the manufacture of dyes (especially sulfur colors), picric acid, picramic acid, diaminophenol hydrochloride (a photographic developer) explosives, and wood preservatives (U.S. EPA/NIH, 1983a; Hawley, 1981). In addition, dinitrophenol (commercial mixture) is used as an indicator and as a reagent for potassium and ammonium ions (Hawley, 1981).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Very few data regarding the fate and transport of 2,3-, 2,5-, 2,6-, 3,4- or 3,5-dinitrophenol in the environment were located in the available literature; however, data that may be useful for predicting the transport and fate of these dinitrophenol isomers in air, water and soil can be estimated from structurally similar nitrophenolic compounds, especially 2-nitrophenol, 4-nitrophenol and 2,4-dinitrophenol.

2.1. AIR

The fate of the dinitrophenol isomers in the atmosphere will depend upon their reactivity with O_3, NO_x and free radicals (e.g., RO·, O· and RO_2·) and their ability to undergo photolysis. It is not possible to predict the fate of the dinitrophenols in the atmosphere since the reactivity of dinitrophenols with atmospheric molecules and free radicals as well as photolytic susceptibility have not been assessed. Furthermore, pertinent data regarding the fate or transport of structurally related compounds in the atmosphere were not located in the available literature.

2.2. WATER

Comprehensive data regarding the environmental fate and transport of the dinitrophenols in aquatic media are not available. The rates of biodegradation of the dinitrophenol isomers in aquatic media have not been determined. However, based on the slight biodegradation of some of the isomers in soil, it is unlikely that biodegradation is a significant factor in determining the fate of these isomers in water.

Data for 4-nitrophenol suggest that it was degraded in aqueous solution within 1-2 months (Callahan et al., 1979). When exposed to sunlight, the principal products formed were hydroquinone and 4-nitrocatechol (Nakagawa and Crosby, 1974). Although no specific information was found demonstrating

that the dinitrophenol isomers would also be photochemically hydroxylated in an analogous manner, they may photochemically degrade under similar conditions. A further photochemical reaction of the dinitrophenol isomers may involve photoreduction of the nitro groups. Nakagawa and Crosby (1974) found that the nitro group of nitrofen, a nitroaromatic pesticide, underwent photochemical reduction to amino and azo groups in an aqueous solution that contained 10% methanol. Aromatic nitro groups are generally reduced photochemically in the presence of suitable hydrogen donors (Morrison, 1969). It is likely that the dinitrophenol isomers may sorb to particulate organic matter containing reducible groups and the organic matter could serve as a reducing agent in the photoreduction. In the absence of other data, it is probable that photodegradation is an important environmental process in determining the fate of the dinitrophenol isomers in both surface waters and soil.

The log octanol-water partition coefficients are 1.75 for 2,5-dinitrophenol, 1.25 for 2,6-dinitrophenol and 2.32 for 3,5-dinitrophenol (Hansch and Anderson, 1967; Leo et al., 1971). The calculated BCFs in trout muscle based on these values are ~12, 8 and 24 for 2,5-, 2,6- and 3,5-dinitrophenol, respectively (Leo et al., 1971), indicating a low degree of bioconcentration for these three isomers. Based on the log octanol-water partition coefficients and on likely complexation with clays, it is likely that 2,5-, 2,6- or 3,5-dinitrophenol will have moderate potential to sorb to organic and clay particles in water media. Therefore, the contribution of leaching to the transport of this compound in the soil will be limited, particularly with soil of high clay or organic content (Callahan et al., 1979). It is likely, however, that the dinitrophenols will enter the aquatic media by runoff.

Data regarding the significance of hydrolysis or oxidation reactions of the dinitrophenol isomers in water or soil were not located in the available literature; however, predictions for nitrophenol hydrolysis, based on hydrolysis data for aci-nitroparaffins, indicate that hydrolysis could occur when the nitrophenols are sorbed to clay particles (Callahan et al., 1979). Specifically, the phenolic group could become more acidic due to the coordination of the phenolic oxygen atom with the metal ions of the clay. This acidic environment could lead to the hydrolysis of the dinitrophenol isomers.

Two processes which influence the transport of the dinitrophenol isomers in water media are volatilization and sorption. Harvey (1959) reported water solubilities (at 35-36°C) of 2200, 680, 420, 2300 and 1600 mg/ℓ for 2,3-, 2,5-, 2,6-, 3,4- and 3,5-dinitrophenol, respectively. These values appear to be highly suspect for two major reasons. First, Harvey (1959) used a crude, dated analytical method to determine these solubilities. Second, water solubilities determined more recently for the structurally related nitrophenolic compounds, 2-nitrophenol, 4-nitrophenol and 2,4-dinitrophenol, are 2100 (at 20°C), 16,000 (at 25°C) and 5600 (at 18°C) mg/ℓ, respectively (Verschueren, 1977). Therefore, assuming that the water solubilities reported by Harvey (1959) are much lower than the actual values, it is likely that the potential for volatilization of any of the dinitrophenols is extremely low. Also, Callahan et al. (1979) reported that data regarding the volatilization potential of nitrophenols in general indicate that these compounds would probably not significantly volatilize from aqueous solutions.

2.3. SOIL

Volatilization and sorption also influence the transport of dinitrophenol isomers in soil. Pertinent data regarding the potential for volatilization of the dinitrophenol isomers from soil were not located, but the

likelihood of volatilization from soil is expected to decrease further if the dinitrophenol isomer is sorbed onto organic matter or clay particles in the soil.

Available data indicate that soil microorganisms may slightly degrade 2,5- and 2,6-dinitrophenol. A low degree of degradability by adapted activated sludge was observed for both 2,5- and 2,6-dinitrophenol when either compound served as the sole source of carbon for the microbes (Pitter, 1976; Pitter et al., 1974).

A number of soil microorganisms adapted to dinitro-o-cresol did not metabolize the nitro group from 2,5- or 2,6-dinitrophenol; only negligible amounts of nitrite appeared with these compounds (Gundersen and Jensen, 1956). One of the organisms in this study was identified as Arthrobacter simplex; other organisms were provisionally referred to as Arthrobacter x and Pseudomonas. An unidentified soluble rust-brown pigment (structure unknown) was observed in the cultures of A. simplex incubated with 2,5- or 2,6-dinitrophenol. It is unclear from the paper if the microorganisms derived any carbon from the compound.

The experimental findings of Chambers et al. (1963) and Tabak et al. (1964) indicate that phenol-adapted bacteria were able to slowly oxidize 2,6-dinitrophenol. The results of these oxygen uptake studies with 2,6-dinitrophenol must be interpreted with caution, as the possibility cannot be excluded that the increased oxygen uptake observed was due to the enhancement of endogenous oxygen uptake by 2,6-dinitrophenol due to its presumed uncoupling effect.

In a comprehensive review of nitroaromatic compounds, Howard et al. (1976) concluded that 2,5- and 2,6-dinitrophenol may persist in the environment for extended periods of time since these isomers appear not to be altered by dinitro-o-cresol-adapted or phenol-adapted cultures.

3. EXPOSURE

No monitoring data on 2,3-, 2,5-, 2,6-, 3,4- or 3,5-dinitrophenol are included in STORET. Games and Hites (1977) monitored the concentration of dinitrophenol (isomer not specified) in the wastewater effluent of a dye manufacturing plant during a 2.5 month compositing study. Dinitrophenol was detected in all eight raw wastewater samples at concentrations ranging from 400-3200 ppb. In the final effluent, 5 of 9 samples contained dinitrophenol at concentrations of 42-2700 ppb. Dinitrophenol was not detected in mud and river water samples collected downstream from the effluent point.

4. PHARMACOKINETICS

4.1. ABSORPTION

Hawley (1981) indicated that dinitrophenol (commercial mixture) is absorbed through the skin; however, quantitative data regarding absorption of the selected dinitrophenol isomers were not located in the available literature.

4.2. DISTRIBUTION

Pertinent data regarding the distribution of the selected dinitrophenol isomers were not located in the available literature.

4.3. METABOLISM

Pertinent data regarding the metabolism of the selected dinitrophenol isomers were not located in the available literature.

4.4. EXCRETION

Harvey (1959) calculated the half-lives of elimination ($t_{1/2}$) for 2,3-, 2,5-, 2,6-, 3,4- and 3,5-dinitrophenol from the blood of mice and rats given a single i.p. dose. The results of this study are given in Tables 4-1 and 4-2.

TABLE 4-1

Elimination Rates of Dinitrophenol Isomers from the Blood of Mice Following a Single Large Dose Given Intraperitoneally[a]

Dinitrophenol Isomer	Dose[b] (mg/kg)	Sampling Times (minutes)	µg Dinitrophenol/g		Half-time of Elimination[c] (minutes)
			Range	Mean ± SD	
2,3-	90	7	33-74	47 ± 16	2.7
	12	8-16	13±3		
2,5-	180	6	33-60	47 ± 13	3.3
	12	9-15	13±4		
2,6-	30	15	49-71	62 ± 8	238.0
	120	28-51	43±10		
	240	10-38	21±11		
3,4-	60	5	38-55	47 ± 9	3.5
	10	9-23	18±6		
3,5-	30	4	44-61	56 ± 11	2.7
	8	5-22	16±8		

[a]Source: Harvey, 1959

[b]Dinitrophenol dissolved in the requisite quantity of sodium bicarbonate or sodium hydroxide.

[c]Half-time of elimination obtained by plotting mean log concentrations against time and reading off time (minutes) for the initial (highest) concentration to be halved.

TABLE 4-2

Elimination Rates of Dinitrophenol Isomers from the Blood of Rats Following a Single Large Dose Given Intraperitoneally[a]

Dinitrophenol Isomer	Dose[b] (mg/kg)	Sampling Times (minutes)	µg Dinitrophenol/g		Half-time of Elimination[c] (minutes)
			Range	Mean ± SD	
2,3-	90	15	70-136	104 ± 22	12.5
	30	31-107	68±32		
	60	4-13	7±3		
2,5-	90	7	8-23	14 ± 6	13.0
	12	8-11	9±2		
	40	2-4	3±1		
2,6-	25	15	64-76	71 ± 10	210.0
	150	36-58	50±9		
	450	10-26	19±9		
3,4-	90	10	41-64	51 ± 11	11.5
	40	1-5	4±2		
3,5-	30	5	22-90	61 ± 30	2.1
	8	10-19	14±4		

[a]Source: Harvey, 1959

[b]Dinitrophenol dissolved in the requisite quantity of sodium bicarbonate or sodium hydroxide.

[c]Half-time of elimination obtained by plotting mean log concentrations against time and reading off time (minutes) for the initial (highest) concentrations to be halved.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of the selected dinitrophenol isomers were not located in the available literature. None of the selected dinitrophenol isomers are scheduled for testing by the NTP (1983).

5.2. MUTAGENICITY

Pertinent data regarding the mutagenicity of the selected dinitrophenol isomers were not located in the available literature.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of the selected dinitrophenol isomers were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of the selected dinitrophenol isomers were not located in the available literature.

5.5. CHRONIC TOXICITY

Pertinent data regarding the chronic or subchronic toxicity of the selected dinitrophenol isomers were not located in the available literature.

5.6. OTHER RELEVANT INFORMATION

The i.p. LD_{50}s for 2,3-, 2,5-, 2,6-, 3,4- and 3,5-dinitrophenol are 190, 150, 38, 98 and 45 mg/kg, respectively, for rats; and 200, 273, 45, 112 and 50 mg/kg, respectively, for mice. The MLD for dogs for 2,3-, 2,5-, 2,6-, 3,4- and 3,5-dinitrophenol are 1000, 100, 50, 500 and 500 mg/kg, respectively (Harvey, 1959).

Burke and Whitehouse (1967) investigated the effect of the dinitrophenol isomers on uncoupling phosphorylation in rat liver mitochondria. They determined a relative potency scheme where 3,5-dinitrophenol > 2,4-dinitrophenol > 2,6-dinitrophenol = 3,4-dinitrophenol > 2,3-dinitrophenol = 2,5-dinitrophenol.

Dinitrophenol (commercial mixture) increased the metabolic rate not only in normal rats but also in rats with induced metabolic acidosis or induced acute uremia (Hohenegger et al., 1982).

2,6-Dinitrophenol is a cataract-forming agent in chickens, inducing the formation of cataracts and death within 5 hours after the oral administration of 0.43 mmol/kg (Buschke, 1947). 2,4-Dinitrophenol has also induced cataracts in the chicken (Buschke, 1947) and in humans who ingested the compound as a weight-reducing drug. Progressive and irreversible cataract formation occurred in humans after months of exposure (U.S. EPA, 1980; Horner, 1942).

3,5-Dinitrophenol was found to inhibit porcine heart malate dehydrogenase *in vitro* (Wedding et al., 1967), while both 2,5- and 2,6-dinitrophenol were reported to inhibit lactate dehydrogenase and hexokinase *in vitro* (Stockdale and Selwyn, 1971).

Several of the dinitrophenol isomers affect the blood system following either *in vivo* or *in vitro* exposures. Motais et al. (1978) reported that both 2,5- and 2,6-dinitrophenol inhibited passive permeability of red blood cells to chloride. 2,6-Dinitrophenol was found to bind to thyroxine-binding sites in human serum albumin (Tabachnick et al., 1970). 2,3-, 2,5-, 2,6- and 3,4-Dinitrophenol produced methemoglobinemia in animals (Magne et al., 1932; Mayer and Vles, 1932).

Grant (1959) reported that the administration of 2,3-, 2,5-, 2,6-, 3,4- and 3,5-dinitrophenol to rats via i.p. injection increased the animals respiratory minute volume (total pulmonary ventilation/minute, determined by multiplying the tidal volume by the respiratory rate in breaths/minute) by increasing both the respiratory rate and the tidal volume. Cameron (1958) also administered the five isomers to rats via i.p. injection at the maximum

non-fatal dose for each isomer, and found that 2,3-, 2,6- and 3,5-dinitrophenol had no effect on the rate of O_2 consumption or on the rate of CO_2 output, while 2,5-dinitrophenol decreased the rate of O_2 consumption and 3,4-dinitrophenol increased the rate of CO_2 output. Levine (1977) observed increased ventilation and oxygen consumption in anesthetized dogs following intra-arterial infusion (3-4 mg/kg) of 2,5- or 2,6-dinitrophenol.

6. AQUATIC TOXICITY

Virtually all of the available information on the acute toxicty of dinitrophenols to aquatic organisms is derived from tests with the 2,4-dinitrophenol isomer, which is not among the isomers discussed in this profile (see U.S. EPA, 1978, 1980).

6.1. ACUTE TOXICITY

Pertinent data regarding the acute toxicity of the selected dinitrophenols were not located in the available literature.

6.2. CHRONIC EFFECTS

The effects of 2,4- and 2,5-dinitrophenol were studied on the survival and development of eggs and embryos of herring, Clupea harengus (Rosenthal and Stelzer, 1970). Concentrations of 2,4-dinitrophenol ranged from 0.92-18.4 mg/ℓ, while 2,5-dinitrophenol was tested at 1.84, 3.68 and 5.52 mg/ℓ. Exposure was from 30, 48 and 55 hours after fertilization until 12 or 13 days in some tests. Percent mortality and malformations of embryos exposed to 2,4- and 2,5-dinitrophenol are summarized in Table 6-1. Although statistical tests were not performed on these data, both 2,4- and 2,5-dinitrophenol appear to increase the mortality and malformations of embryos at levels near 1-2 mg/ℓ. Malformations included curled bodies, undifferentiation, and unilateral and complete anophthalmia.

6.3. PLANT EFFECTS

Pertinent data regarding the effects of the selected dinitrophenols in aquatic plants were not located in the available literature.

TABLE 6-1

Percent Mortality and Malformations in *Clupea harengus*[a]

Chemical	Concentration (mg/ℓ)	Mortality (%)	Malformations (%)	Number of Exposed Embryos
None (control)	0.0	26.6	5.5	111
2,4-Dinitrophenol[b]	0.92	46.9	25.0	32
	1.84	58.6	24.1	29
	2.76	56.5	16.3	43
	3.68	65.8	18.4	38
	4.60	70.0	14.8	27
	5.52	100.0	NA	49
None (control)	0.0	19.7	2.1	48
2,5-Dinitrophenol[c]	1.84	34.5	5.4	55
	3.68	61.1	5.5	36
	5.52	66.0	16.1	47

[a]Source: Rosenthal and Stelzer, 1970

[b]Exposure was from 48 hours after fertilization until 12 days.

[c]Exposure was from 55 hours after fertilization until 12 days.

NA = Not applicable

6.4. RESIDUES

Pertinent data regarding residues of dinitrophenols in aquatic organisms were not located in the available literature. However, as discussed in Section 2.2., calculated BCFs based on octanol-water partitioning indicate no significant potential for bioconcentration of these compounds in aquatic organisms.

6.5. OTHER RELEVANT INFORMATION

Additional information on the toxic effects of the selected dinitrophenols on aquatic organisms were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

The U.S. EPA (1980) has set ambient water quality criteria for the protection of human health from dinitrophenols (all isomers) at 70 µg/ℓ for ingestion of water and aquatic organisms, and 14.3 mg/ℓ for ingestion of aquatic organisms alone. These criteria are based on the toxicity of 2,4-dinitrophenol and, in particular, on its cataractogenic properties in humans.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of the selected dinitrophenols were not located in the available literature. Due to the lack of appropriate chronic data on aquatic toxicity, the U.S. EPA (1980) did not suggest criteria for aquatic life for any nitrophenols.

8. RISK ASSESSMENT

The lack of carcinogenicity and chronic or subchronic toxicity assays precludes quantitative risk assessment for any of the selected dinitrophenol isomers. Data regarding the mutagenicity, teratogenicity or other reproductive effects of the selected dinitrophenols were not available. Acute effects of the dinitrophenol isomers on animals include uncoupling oxidative phosphorylation (Burke and Whitehouse, 1967), cataract formation (Buschke, 1947), enzyme inhibition (Wedding et al., 1967; Stockdale and Selwyn, 1971), blood disorder (Motais et al., 1978; Tabachnick et al., 1970; Magne et al., 1932; Mayer and Vles, 1932), and changes in respiration (Grant, 1959; Cameron, 1958; Levine, 1977).

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APPENDIX: LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

These searches were conducted in May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

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